More to Atherosclerosis Than Stenosis
Symptomatic Carotid Artery With Intraplaque Hemorrhage

Samhitha Rai, MD; David E. Thaler, MD, PhD; Payam Salehi, MD, PhD; Neel Madan, MD; Lester Y. Leung, MD

Case Description
A 66-year-old man presented with left facial weakness on awakening. He had cerebrovascular risk factors, including HIV, hypertension, and hyperlipidemia. When the facial weakness did not resolve after a few days, he presented to a local hospital. A brain magnetic resonance imaging scan, including an axial diffusion-weighted imaging sequence, demonstrated an ischemic stroke in the face representation area of the right precentral gyrus. Transthoracic echocardiography, carotid ultrasound, and cardiac telemetry were unrevealing. He was prescribed dual antiplatelet therapy and a statin. He was referred to a vascular neurologist at our tertiary care center. After seeing the vascular neurologist, a noncontrast magnetic resonance angiogram (MRA) of the head and neck was obtained: 3-dimensional (3D) time-of-flight (TOF) imaging demonstrated ≤50% stenosis of the carotid bulbs bilaterally (40%–50% bilaterally by NASCET criteria [North American Symptomatic Carotid Endarterectomy Trial]). On these T1-weighted images and on a fat-suppressed axial T1 sequence, there were focal, crescentic, subintimal hyperintense signals at the bifurcations bilaterally (Figure 1). The findings were highly suggestive of intraplaque hemorrhage. However, given the modest degree of stenosis and an incomplete etiologic investigation, the right carotid artery was not yet considered definitively symptomatic. He then underwent 30 days of extended cardiac rhythm monitoring to investigate occult arrhythmias as a potential source of the presumed embolic stroke. The facial weakness completely resolved.

Two months later, he developed recurrent left facial weakness accompanied by new left arm weakness and dysarthria. When he presented to the local hospital, his National Institutes of Health Stroke Scale (NIHSS) was 5. He was not treated with intravenous tPA (tissue-type plasminogen activator) because of a delay in presentation. A brain magnetic resonance imaging scan, including an axial diffusion-weighted imaging sequence, demonstrated a similar degree of stenosis at both carotid bifurcations but <50% stenosis.

A carotid ultrasound was performed to better visualize plaque morphology. It revealed an irregular, calcified plaque present in the right internal carotid artery with 16% to 49% stenosis. A transesophageal echocardiogram revealed only mild atherosclerotic plaque in the aortic arch and descending aorta. His extended cardiac rhythm monitor had not detected any arrhythmias.

Despite only demonstrating a modest degree of internal carotid artery stenosis, the right carotid lesion was thought to be the source of emboli. Vascular surgery was consulted to evaluate for possible carotid endarterectomy. After careful deliberation between neurology, vascular surgery, and neuroradiology, a carotid endarterectomy was performed. Intraoperatively, the vascular lesion was identified as a round, dark red protrusion on the outside of the right proximal internal carotid artery (ICA). The resected lesion was found to be an ulcerated, ruptured, hemorrhagic plaque (Figure 3).

The patient was discharged to a rehabilitation facility on antiplatelet and high-dose statin therapy.

Discussion
We present a patient with an embolic ischemic stroke and early stroke recurrence in the setting of extracranial large artery atherosclerosis with high-risk morphological features but <50% stenosis.

Current American Heart Association/American Stroke Association (AHA/ASA) guidelines only address the degree of arterial narrowing (% stenosis) when recommending the use of carotid revascularization procedures for secondary
stroke prevention. This guideline is largely informed by the NASCET, VACS (Veterans Affairs Cooperative Study), and ECST (European Carotid Surgery Trial) trials that found benefit for carotid endarterectomy in patients with a symptomatic carotid stenosis of ≥70%. The most recent AHA/ASA guideline advises against revascularization procedures in patients with <50% extracranial ICA stenosis. In NASCET, the arm of the trial assessing efficacy in patients with symptomatic carotid stenosis of 30% to 69% did not demonstrate clear benefit. This group was later subdivided, showing modest benefit for surgery with 50% to 69% stenosis. These guidelines and clinical trials do not account for plaque morphology that may predict a high risk of early stroke recurrence as demonstrated by our patient.

Given this patient’s HIV status, one may wonder if this disease played a role in his atherosclerotic plaque morphology. Although there is evidence from previous studies to indicate an association between HIV and large artery atherosclerosis in patients with ischemic stroke, a more recent study suggests...
that the main factors that independently increase risk of stroke for people with HIV are immunosuppression (CD4+ cell count <500/µL) and viremia (HIV RNA >500/mL), after adjusting for other vascular risk factors. Our patient was immunocompetent with an undetectable viral count, making HIV a less likely contributor in his case.5

Because of its noninvasive nature and low cost, ultrasonography remains a widely used modality for the evaluation of carotid stenosis. Recent studies have looked at advances in ultrasound technique to assess plaque morphology as a predictor of plaque stability, independent of the degree of stenosis. In a study using ultrasound digital imaging analysis software to assess plaque echodensity and texture, echolucency and homogeneity were associated with unstable histological phenotypes.6 These techniques have largely been limited to research settings and are not used in routine clinical practice.

Computed tomography angiography and MRA can identify atherosclerotic lesions in the carotid arteries with high levels of accuracy, sensitivity, and specificity. Computed tomography angiography can discriminate between lipid components, fibrous components, and the calcium present in atherosomas.7 T1-weighted images on magnetic resonance imaging can help to identify subintimal hemorrhage. Accordingly, both modalities can be helpful in assessing clinically relevant morphological features of extracranial carotid plaques.8 Carotid intraplaque hemorrhage may be an independent risk factor for recurrent cerebral ischemia and may identify those that would benefit from early surgical intervention.9

However, as demonstrated in this case, the sensitivity and specificity can vary depending on the imaging sequence (and the scanner) used. A 3D TOF sequence will be far superior to a 2D TOF sequence. Axial T1 fat-suppressed images can be complementary on all of these scanners. Moreover, the findings on a postcontrast MRA neck sequence are only apparent if the noncontrast images used for subtraction of the MRA are reviewed. Thus, it is important to be aware of local variations in practice, and how intraplaque hemorrhage will appear on the sequence used in the carotid evaluation.

Patients may require advanced or multimodal imaging for optimal assessment of their atherosclerotic plaques. Our patient presented with recurrent stroke secondary to symptomatic carotid disease that did not meet criteria for carotid endarterectomy following current guidelines. Ultrasonography did not detect the high-risk morphological features of the ICA plaque in this patient. Combining the intraplaque hemorrhage on MRA and the clinical history, we referred the patient for surgical intervention based on clinical judgment. This case demonstrates the importance of considering surgical intervention when plaque morphology is suggestive of a high-risk extracranial ICA lesion as the source of emboli. Plaques can be considered embolically significant even though hemodynamically they are not.

**TAKE-HOME POINTS**

- Although clinical trials to date have not addressed atherosclerotic plaque morphology, this should be considered when determining the source of cerebral or retinal emboli (especially in the setting of failing medical therapy) and when selecting patients for revascularization procedures.
- Computed tomography angiography and magnetic resonance angiogram are essential, and potentially complementary, imaging modalities for assessing the extracranial and intracranial vasculature for culprit lesions. Variations in magnetic resonance angiogram neck technique need to be understood to be able to maximize diagnosis of potentially vulnerable plaques. Carotid ultrasonography is less sensitive for detecting clinically relevant morphological plaque features.

**Disclosures**

None.

**References**


KEY WORDS: atherosclerosis ◼ carotid stenosis ◼ dysarthria ◼ hypertension ◼ stroke

Downloaded from http://stroke.ahajournals.org/ by guest on April 1, 2017
More to Atherosclerosis Than Stenosis: Symptomatic Carotid Artery With Intraplaque Hemorrhage

Samhitha Rai, David E. Thaler, Payam Salehi, Neel Madan and Lester Y. Leung

Stroke. 2017;48:e104-e107; originally published online March 14, 2017;
doi: 10.1161/STROKEAHA.117.016923

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2017 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/48/4/e104

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/